Society Proceedings

NEW YORK NEUROLOGICAL SOCIETY.

January 2, 1906.

The President, Dr. Joseph Fraenkel, in the Chair.

A Case of Neurasthenia, with Exophthalmos.—Presented by Dr. William M. Leszynsky. The patient was a woman, thirty-five years old, who was sent to Dr. Leszynsky two years previously with the diagnosis of Graves' disease. After examination, Dr. Leszynsky concluded that the diagnosis had been made solely upon the marked protrusion of the eyeballs, as the other classical symptoms of Graves' disease were entirely lacking. Her general symptoms were of the neurasthenic type, and the exophthalmos was due to a high degree of myopia. The patient's vision was much impaired. The pulse rate ranged between 72 and 74 per minute.

The President, Dr. Fraenkel, said the older writers had distinguished between two forms of protrusion of the eyeball; one they called exophthalmos, and the other buphthalmos, under which title they included a permanent enlargement of the entire eyeball.

Report of Two Cases of Polioencephalitis, with Autopsies.—By Dr. Nathaniel Bowditch Potter. In connection with the report of these cases, Dr. Potter showed a third case, a colored woman, forty-one years old, a laundress, who was admitted to the New York Hospital in February, 1901. She gave a history of having had four miscarriages, and one child that survived three months. Denied syphilis, secondaries, and the use of alcoholic stimulants. She was brought to the hospital in an ambulance, having been found on the street in a comatose condition, with a bottle containing about two ounces of what was probably wood alcohol in her possession. Her stomach was emptied, and the washing smelled strongly of the same material. The breath also contained a strong odor of wood alcohol. The pupils were unequal, the right somewhat contracted; the left moderately dilated. The urine contained a heavy trace of albumin, numerous granular casts, many pus and epithelial cells. After washing out the stomach, the patient could be partly aroused. Slight impairment of power on the right side was noticed. She gradually improved, and left the hospital on March 3, 1901.

The patient was re-admitted to the hospital on Sept. 20, 1905, with the following history: Two days before admission, while working, she suddenly began to feel "funny," as though her legs could not support her, and then sank down on the floor. She did not lose consciousness, and in a few moments got up and went to bed. There was neither pain nor weakness, but on attempting to walk, she found that she could not do so without assistance. There was considerable mental confusion, which had persisted.

There was slight asymmetry of the face. The left pupil reacted slightly and rather sluggishly; the right reacted promptly; there was slight nystagmus. She had an ataxic gait. The knee jerks were exaggerated, the right more so than the left; no ankle clonus; no Babinski; no impairment of sensation. This case, Dr. Potter said, was apparently one of polioencephalitis of the Wernicke type. In addition to the methyl alcohol poisoning as a cause in this case, it was interesting to know that the patient had probably had syphilis.

Dr. Conner sent the patient to the City Hospital, where she was admitted on Oct. 25, 1905. Her mental condition gradually improved up to the present, and now she is reasonably intelligent in regard to time and place. She has been much less ataxic than is evident at present. The effort of the trip from the City Hospital has fatigued her somewhat. She exhibits a smooth atrophy of the base of the tongue.

Two other cases of polioencephalitis were reported by Dr. Potter. Both of these were observed at the City Hospital during the past eighteen months, and in both, autopsies were obtained. The first case was that of a married woman, forty years old, a native of Hungary, who was admitted to Dr. Child's service at the City Hospital on Sept. 20, 1904. She had four children, the first twelve years ago; the last five years ago. There was a history of one miscarriage, six years ago. The patient was addicted to the moderate use of alcohol.

Three weeks before admission she began to complain of headaches, dizziness and vomiting. When she was first seen by Dr. Potter, on Sept. 23, 1904, she was in a semi-comatose condition, and resisted interference, especially on moving the head. She was well nourished and fairly muscular. The pupils were equally dilated, and reacted sluggishly to light and accommodation. The teeth were covered with sordes, the breath was foul and somewhat urinous. The pulse was rapid and regular, with slightly increased tension. The reflexes were much diminished. Kernig's sign was present; more marked on the right side; Babinski absent. The urine, with a specific gravity of 1019, contained a trace of albumin, and a few hyaline casts. By lumbar puncture, two drams of clear serum were withdrawn under no pressure; this fluid contained a few cellular elements, but no organisms were found. On Sept. 26, the patient was brighter; she could readily be persuaded to show her tongue, and the contracture of the right eyelids had vanished. There was a slight, paralytic ptosis of the left eye. Kernig's sign was absent. The pupils were dilated, the left being larger than the right; they failed to react to light. There was slight flaccidity of the right side of the face. The reflexes of the lower extremities were absent. The plantar reflexes were flexor in type. On Sept. 27 the patient was more comatose, and she died on the following day.

Autopsy: The meninges were intensely congested, but the brain surface was dry. The base of the brain was moderately edematous. The pia was deeply injected, and distinctly hemorrhagic and cloudy in places. The brain surface, as a whole, was rather pale and firm. The vessels were firm, and apparently numerous thrombi were diffusely distributed through the gray and white matter. The color of the cortex varied; it was mostly cloudy and gray, but frequently a diffused grayish pink or even a decided red. The basal ganglia anteriorly were cloudy gray, but not hemorrhagic; posteriorly, there were frequent thrombi with large perivascular spaces. In the pons and fourth ventricle the vessels presented the same condition. The cerebellum was in the same condition as the cerebrum. There was no edema of the spinal cord. The pia was injected, the superficial vessels being prominent and rather soft. In the cervical region, the gray matter was almost liquefied, of a pinkish-gray color, with a deep injection of the vessels. In the dorsal region the gray matter was more distinctly pinkish. In the upper lumbar region the gray matter was almost entirely liquefied, and intensely hemorrhagic.

The second case reported by Dr. Potter, for which he was indebted to Dr. Evan Evans, was a male, an Italian, thirty-seven years old, married, who was admitted to the City Hospital on Sept. 8, 1904. His family history was negative. His occupation was that of a driver. He denied intemperance. In October, 1903, he had a local venereal sore, which lasted one month. There was no history of rash, alopecia or sore throat. The patient stated that this lesion appeared one month after his last coitus. He consulted a physician, who prescribed pills, which the patient took for twenty-five days. He had never had malaria.

The patient stated that in June, 1904, he first began to suffer with pains in the legs, and dizziness on standing or walking. No pulmonary symptoms nor night-sweats. No marked disturbance of vision; no pain in abdomen. His difficulty in walking was not increased by darkness; there was no paresthesia.

A physical examination of the patient on admission showed a fairly well nourished man. There was a fine maculopapular rash over the body and legs, and an old bubo scar in the groin. The pupils of both eyes were equally dilated; the left responded sluggishly to light, but was normal to accommodation; the right responded to accommodation, but not to light. There was a marked tremor of the tongue; no deviation.

The patient died on Sept. 16, 1904. The autopsy showed the case to be one of acute encephalitis, with a general ascending poliomyelitis. There was considerable edema of the brain, and the gray matter of the cord, as in the preceding case, was soft, pinkish and hemorrhagic.

In both of these cases, microscopic sections showed in the meninges marked congestion, and presented a turbid, hazy appearance, due to inflammatory edema. The vessels were enormously congested, and frequently surrounded by cellular infiltration, partly localized, partly progressing in the lymph and tissue spaces to a more diffuse distribution. The vessel walls showed numerous areas of hyaline thickening. In the neighborhood of some of these vessels, a collection of more elongated nuclei, apparently derived from the fixed tissue elements, was visible. Nuclear remnants in other parts, especially where the meningeal tissue had lost its entire structure, were frequently seen. The brain substance presented a diffusely hazy appearance, with the ganglion cells throughout extensively degenerated; the protoplasm poor in granulation, pale pigment, however, having been retained in a large number. The neuroglia was hazy; free round cell nuclei were diffusely distributed, and the vessels were enormously engorged, sometimes containing a large number of leucocytes, which, in places, lay outside the vessel wall. The perivascular spaces were wide. As a whole, there existed no marked old thickening of these vessels. In places, an active softening and loss of brain tissue could be seen.

Sections of the cord showed practically the same intense inflammatory lesion throughout. The vessels more frequently showed old hyaline thickening. The destruction of the white matter was quite irregular throughout, and did not show any localized involvement of definite tracts.

An examination of the rest of the organs in the body showed acute parenchymatous degeneration, and the kidneys presented an acute hemorrhagic nephritis.

The autopsies and sections were made and reported upon by Dr. Horst Oertel, pathologist to the City Hospital.

Dr. J. Ramsay Hunt said the woman showed by Dr. Potter had been seen by him some months ago at the New York Hospital. Previous to that time the patient had been examined in the very acute stage by Dr. Ward A. Holden, who had made the diagnosis of polioencephalitis superior. When Dr. Hunt saw the patient, she was somnolent and much confused mentally. There was marked ataxia, of the cerebellar type; the intrinsic muscles of the eye were normal, and the reactions were fairly prompt. There was no ptosis, but some weakness of the internal and superior recti on both sides, and the ocular excursions were accompanied by coarse nystagmus.

Dr. Hunt said he had seen two other cases of polioencephalitis at about the same stage as this woman was in, and he emphasized the importance of the recognition of the condition, in order to avoid confusion with other cerebral affections, especially syphilis. In both cases, weakness of the extrinsic muscles of the eye, diplopia, nystagmus and cerebellar ataxia characterized the clinical picture, following prolonged alcoholic debauches and delirium.

Six months ago, Dr. Hunt said, he saw a typical case of polioencephalitis of the Wernicke type. This he was able to follow to the autopsv table, and he had reported the case in full at the last meeting of the American Neurological Association. The onset was acute, with headaches, delirium, ataxia, and paralysis of the third and fourth cranial nerves. Later, bulbar symptoms were superadded, the patient dying on the fifth day. The brain was subjected to a careful histological study, with numerous sections through the medulla, pons and thalamic regions, and the only lesions found were microscopic hemorrhages and small collections of round cells, both in the pons and the medulla.

The lesions in these cases, Dr. Hunt said, were not restricted to the gray matter. In his original description, Wernicke regarded the condition as a polioencephalitis, but of some thirty cases, with histological studies, which the speaker said he had been able to collect in literature, almost one-third showed disseminated lesions both in the gray and white matter, so that it was more proper to regard these cases as a disseminated encephalitis. Cases of this type (Wernicke) also presented combination forms and transition states to the polioencephalitis of the Strümpell-Leichtenstern type, and encephalitis in general.

Dr. Harlow Brooks said that some years ago he reported a case similar to the one referred to by Dr. Hunt. The lesions were not confined to the gray matter, but were also in the white matter. The etiological factor in this case, Dr. Brooks said, was apparently some coal tar product, probably antikamnia. The case was a rapidly fatal one, and the symptoms were rather more acute than in the case shown by Dr. Potter. Otherwise, it was practically the same.

Dr. Potter said he had shown and reported these cases on account of the comparative rarety of the condition, and the interest that attached thereto. He inquired as to the importance of syphilis as an etiological factor. In many of the cases where alcoholism had been assigned as the causative factor, there was a preceding history of syphilis.

Dr. Hunt said the consensus of opinion seemed to be that alcohol was the commonest etiological factor in these cases. While there was often a history of syphilis, the connection between that disease and polioencephalitis was not as distinct as it was to alcoholism. There were also other factors which apparently bore some relation to it, but they were more or less obscure, and suggested a toxemia.

An Analysis and Report of Thirty Cases of Epidemic Cerebrospinal Meningitis.—Dr. William M. Leszynsky read a paper on this subject, and presented eight of the cases, with a brief history of each. Of the thirty cases reported, fifteen had recovered. Among the cases shown there was one boy who had lost the sight of one eye through an iridochoroiditis, and another who became totally and permanently deaf on the second day of the disease. In the other cases shown, the recovery was apparently complete.

Dr. Edward D. Fisher said that most of the cases of epidemic cerebrospinal meningitis that had come under his observation had been in patients who were older than the cases reported by Dr. Leszynsky. He had seen very few cases in children. The treatment was practically limited to good nourishment, spinal tapping, and, in some cases, the administration of the salicylate of sodium.

As regarded the prognosis of the disease, Dr. Fisher said it should be made with great caution, and the seriousness of the case could not always be judged by the eye symptoms. The most serious cases sometimes recovered, while the apparently mild ones not infrequently died. The prognosis improved with the abatement of the epidemic.

In speaking of protracted cases, Dr. Fisher referred to one of almost nine months' duration. In that case, convulsions were a prominent feature. The patient was apparently on the road to recovery, when he had a third relapse and died.

Dr. Arthur C. Brush, of Brooklyn, N. Y., said he had seen many cases of cerebrospinal meningitis during the recent epidemic, and he could confirm all that Dr. Leszynsky had said in regard to the erratic course of the disease. At times, the most desperate cases recovered, while the apparently hopeful ones suddenly died. The treatment, so far as he was concerned, was entirely symptomatic.

Dr. Brush said he was surprised to learn that deafness had resulted in so few of Dr. Leszynsky's cases. In his own experience the percentage of deafness was much higher. Another possible sequela that should be borne in mind was epilepsy, coming on a year or two after the termination of the disease.

Dr. Harlow Brooks said he had long regarded cerebrospinal meningitis as a neurological disease, and it appeared from the number of cases reported by Dr. Leszynsky that the neurologist was at last coming into his own. The results given by the reader of the paper were decidedly more favorable than we were accustomed to look for in dealing with these epidemic cases, and it was a very impressing demonstration to see so many cases recover without serious sequelæ.

Dr. Brooks said that an important contribution to the pathogenesis of cerebrospinal meningitis had recently been made by Dr. E. K. Dunham, of the Meningitis Commission, which might eventually lead to a more enlightened method of treating the disease. A chemical substance had been discovered to be formed by the meningococcus, which belonged to the hemorrhagins. This causes a solution of the endothelial cells in the walls of the blood vessels, and was supposed to be responsible for the enormous exudates that were so characteristic of the early stages of cerebrospinal meningitis.

Dr. Edwin G. Zabriskie said that actuated by an article by Tretrop, in the *Annals of Otology, Rhinology and Laryngology,* March, 1905, on the results of treatment in cases of deafness, vertigo and tinnitus by the Babinski method, several surgeons at the Manhattan Eye and Ear Hospital had requested lumbar puncture for bilateral deafness following cerebrospinal meningitis. Lumbar punctures were made at intervals of from five to ten days on about twelve cases, without any relief as regarded the deafness.

Dr. Leszynsky, in closing, said that lumbar puncture could not be expected to affect the deafness in these cases, as it had been demonstrated in fatal cases that the loss of hearing was due to infiltration and complete destruction of the aural nerve. In a few instances, perhaps, it was due to inflammation of the middle ear.

The following officers were elected for the ensuing year: President, Dr. Joseph Fraenkel; First Vice-President, Dr. Adolf Meyer; Second Vice-President, Dr. J. Ramsay Hunt; Recording Secretary, Dr. Edwin G. Zabriskie; Treasurer, Dr. G. M. Hammond; Corresponding Secretary, Dr. F. K. Hallock; Councillors, Dr. M. Allen Starr, Dr. Charles L. Dana, Dr. Joseph Collins; Dr. J. Arthur Booth and Dr. William M. Leszynsky.

February 6, 1906.

The President, DR. JOSEPH FRAENKEL, in the Chair.

Case of Carcinosis of the Brain and Cord.—Dr. Walter Timme reported this case, and showed the specimen. The patient was a married woman, who was forty-three years old at the time of her death. She was the mother of several healthy living children. In 1899 she developed an ulcer of the leg, which resisted all forms of treatment for about a year, and then healed within a few weeks under dram doses of potassium iodide. Syphilis was denied. In 1901 she was operated on by Robert Abbe for a typical scirrhus carcinoma of the left breast, and the breast, pectorals and axillary lymphatics were removed. In October, 1903, two years after the operation, there were no evidences of a recurrence in the scar or elsewhere, and the patient was apparently in excellent health, with the exception of neuralgic pains at the site of the right fifth and sixth intercostal posterior roots, and in the side. At this time, the diagnosis of spinal metastasis of the dura and nerve roots was made by Dr. Abbe, and it was necessary to begin the use of morphine to relieve the pain. The patient was not bedridden, but the pain was very severe, and the use of morphine and anodynes was constantly necessary.

During the year 1904, one consultant after another was called in, and none agreed with the original diagnosis of spinal metastasis. The diagnosis was variously made as pure neuralgia, neuritis, hysteria, neurasthenia, or a combination of these latter conditions. The patient developed a slight convergent squint, which was accentuated after the administration of morphine. X-ray and radium treatment were given a trial, without any benefit. Morphine was constantly resorted to, although in small doses.

The patient was first seen by Dr. Timme on Sept. 2, 1905. Her general condition was good. Her facial expression gave one the impression that she was suffering real physical pain, and this was the only symptom for which she asked relief. This pain was in the nature of an intense rachialgia at the base of the spine, thence radiating for a short distance up the spine, and downward along the course of both sciatic nerves. She complained of practically nothing else. A minute after a hypodermic of morphine was administered she was smiling and free from pain. There was no headache; no vertigo; no nausea; no vomiting. The appetite was fair; the mental condition excellent. There was an area of alopecia on the left side of the head, about the size of a silver half dollar. This was subsequently found to correspond exactly with the position of one of the tumors lying directly underneath that part of the vault. The skin of the entire body had imbedded in it discrete, non-inflammatory masses, about the size of a hazel nut. The scar of the operation was in perfect condition, and no glandular enlargements could be made out. The heart, lungs and other organs seemed normal. An examination of the urine showed a large excess of phosphates, but was otherwise negative. The entire spine was hypersensitive, and at the level of the fifth, sixth and seventh vertebræ, pressure elicited shrieks of pain. This symptom remained constant. There was a band of anesthesia around the abdomen, varying in width and position daily. On either side of this band was an area of paresthesia. There was no loss of knee jerks. Both pupils were contracted, though they were not of the Argyll-Robertson type. The left eye showed some nystagmus. There was ptosis of the left lid, and a slight convergent squint. The tongue deviated markedly to the left, though the patient could correct this somewhat upon trial. The temperature of the right hand was always two degrees higher than that of the left. Almost all the usual symptoms of hysteria were ingrafted upon the foregoing; thus, the globus, the insensitive corneæ, the craving for sympathy, the hysterical crying were all present. There was slight thickness and slowness of speech. Deglutition was also slow, and necessitated effort.

The patient was given a vigorous course of anti-syphilitic treatment, without any improvement. Early in October, 1905, the morphine was discontinued. The patient, after the first few days of extreme agony following its deprivation, began to show signs of improvement. She complained less of her backache, and appeared brighter. However, her speech became somewhat thicker, and deglutition was more difficult. On Oct. 14 her breathing became labored, and the temperature rose to 103. By the following morning, cyanosis had developed, and the patient could now be aroused with difficulty. The respirations rose to 72 per minute, and the rectal temperature to 107, 108, and finally to 109.5. The pulse rose to 150 per minute; edema of the lungs set in, and death soon supervened.

The autopsy showed secondary general carcinosis of the brain, spinal cord, lungs, ovary, skin and vertebræ. In the brain there was a tumor on the inner surface of the dura, pressing upon the left cortex. It was about an inch and a quarter in diameter. It was located above the Sylvian fissure, and rested on the posterior, upper part of the inferior frontal, and the lower, posterior part of the middle frontal, and on the inferior part of the post-central and the anterior central gyri. A larger tumor, two and a half inches in diameter, was found on the base of the brain, extending from the left frontal region posteriorly to the orbital plate. This tumor had involved the bone in the immediate vicinity. Both tumor masses had indented the brain tissue by their constant growth. The spinal cord, from the fourth cervical vertebra to the cauda equina, was notably compressed by a metastatic growth extending along the dura mater spinalis. The vertebræ, in several places, were infiltrated and spongy. The nerve roots were involved by carcinomatous infiltration, especially in the upper dorsal region. The microscopical examination of the various metastatic growths showed carcinomatous characteristics.

In concluding the history of this case, Dr. Timme called attention to the fact that although this patient had been examined by five or six competent neurologists during the past two years, the diagnosis they made varied from "pure hysteria" to "neuritis based upon morphinism," and in only a single instance did the opinion come near the truth. When Dr. Joseph Fraenkel saw her in consultation about two years ago he expressed the opinion that she was suffering from a metastatic growth of the cord. His diagnosis was arrived at by exclusion, and nothing was said of any possible brain involvement. In view of the extensive interference with the cerebral cortex, one could but wonder at the paucity of signs and symptoms presented during life.

Dr. Edward D. Fisher, in discussing Dr. Timme's case, said that so far as the growth in the frontal lobe was concerned, it could be well understood that a tumor might exist in that area without causing any mental or physical disturbance. In regard to the second tumor in the motor area there are many cases on record where large growths in that region have given rise to few or no symptoms. He recalled one case of that character, where the presence of the tumor was verified by operation, in which there were no motor symptoms, and even optic neuritis was absent.

The spinal symptoms mentioned by Dr. Timme were simply the effects of pressure, and should have led one to suspect the presence of a malignant growth involving the cord.

Dr. Fisher said he was entirely in accord with the suggestion made by Dr. Timme that a consulting physician should not be expected to base his diagnosis upon a single examination.

The President, Dr. Fraenkel, said that with the better knowledge of neurology, the diagnosis hysteria was becoming less frequent.

In the case reported by Dr. Timme, Dr. Fraenkel said he had made the diagnosis of a metastatic growth of the cord three years before the patient's death. The chief symptom upon which he had based the diagnosis was the permanent neuralgia, which was in the strict anatomic distribution of an intercostal nerve. Further, on the fact that the organic nature of the neuralgia was proven by a border of anesthesia above and below this nerve. This, in conjunction with the history of the removal of a carcinoma from the breast, made the diagnosis relatively easy.

Discussion of the Data Concerning Neuro-Fibrils, with Illustrations.—Dr. Adolf Meyer, at the request of the President, gave an illustrative demonstration of the data present in the literature concerning the problem of neuro-fibrils. It forms the most prominent issue of the histological side of neurology, and while not as fascinating as the matters which are more directly accessible to experiment, it is an issue worth careful review to prevent the tendency to draw speculative conclusions in a field in which the microscopist is so easily influenced by what he wishes to see. It is indeed impossible to understand the discussion of the neuro-fibrils without due consideration of the setting in which the orthodox theories of neuro-fibrils of Apàthy, Bethe and Nissl arose. There is no doubt that the neurone speculation came at a time when the cell-concept and the ideas concerning syncytia and cell-bridges and intercellular substances had a special fascination, as is shown in Weigert's attitude concerning neuroglia.

Dr. Meyer pointed to the remarkable findings of Deiters, to the diffuse net of Golgi, which was ignored by most workers with his method in favor of the simpler tree-simile, then the return to the study of the nerve-cell protoplasm, the discovery of fibrils by Becker, and the gradual development of Bethe's views and their relation to Apáthy. The demonstration of non-anastomosing fibrils in the majority of cell bodies and processes and the existence of nets in the spinal ganglion cells, and a few other types; the probable independence of the fibrils, their isolation in the Ranvier rings, the demonstration of primary stainability of the fibrils, the gradual shifting in the interpretation of the relation of the Golgi net to the extra-cellular and intra-cellular fibrils. With Bethe's results he contrasts those of Donaggio, with a fibril net prevading the whole cell-body and fibril bundles in the dendrites, and the importance attached to the cell-body as a place of reception and synthesis of stimuli. Rossi's pictures are referred to as showing wholly incomparable meshes throughout the cell-body; Bielschowsky, as showing conditions more nearly identical with Bethe's results, but also with rather frequent perinuclear net-work; and Cajal, as giving fibrils and nets, and especially clear, a wonderful system of end-feet of the fibrils terminating on the cells.

This was followed by a review of the history of Held's end-feet, of the Golgi net, of the terminal nerve-net, and the controversy concerning the transition of fibrils from the end-feet into the cell-body. It was shown how much the pictures furnished depend on the general histological views of the writers, and how fallacious it would be to attribute too much importance to these, as yet, conflicting data. Schaffer's generalizations from his findings in amaurotic idiocy are added as an additional instance of the determination of the views of the writers by previous work or special observations. The conflicting ideas concerning the embryological origin of fibrils was next brought out, and also the very conflicting statements in such a clear cell-alteration as the axonal alteration in the descriptions of Bethe, of Donaggio and Marinesco and Bielschowsky. For the time there seems to be no evidence that the fibrils are anything but a special adaptation of the local protoplasm. The idea of fibrils "going in" is hardly supported. Where there are changes in the fibrils there are almost invariably other changes in the local protoplasm. The method of Bielschowsky is probably the most useful for routine work and comes nearest to the histologically most convincing pictures of Bethe; and that it gives valuable data of anomalies of structure not observed with other methods was to be shown in the demonstration of specimens by Dr. Lambert.

Dr. C. I. Lambert, presenting the neuro-pathological application of the several fibril methods, gave a comparative résumé of the ones most adapted to this kind of work.

Donaggio's method is not to be especially recommended. Dugaro's substitution of colloidal silver for Joris' gold colloid promises little more. However, his axis-cylinder method with Bethe's blue or Mallory's iron-hematoxylin method gives very satisfactory results. More dependence is to be placed upon the silver methods of Cajal and Bielschowsky, which are more certain, uniform, and more nearly identical in their respective cell renderings. Cajal's method, with its several modifications, although usable in pathological conditions, is essentially a method for normal histology. The uncertain character of *en masse* impregnation, the lack of uniformity in the individual nerve cell reactions, even under normal conditions, do not permit of equally trustworthy comparative work under anomalous conditions, where too frequently there is a minimum amount of available material, often improperly fixed. However, the successful Cajal picture is full of refined detail, and for normal cell morphology is par excellent. On the other hand, the frozen section methods of Bielschowsky for cell and fibre studies lend themselves readily to the neuro-pathologist.

The directness of the method, the comparative ease in manipulation and control, the uniformity under comparable conditions, recommend its use where more intimate details and a fibril equivalent is desired.

About thirty lantern slides were shown illustrating cell types after Cajal's and Bielschowsky's methods in normal and pathological conditions.

Briefly summarized, in general paralysis, the cortex-width and lamination, and the general and focal disturbances, are easily demonstrable by these methods, especially Bielschowsky's. The form and intrinsic nerve cell structure, and its relative integrity, is exceptionally well rendered. Swelling of the interfibrillary substance, with more or less gluing together of the fibrils, apparent fragmentation and granulation of the intra-cellular neuro-fibrils, varied by a frequent vacuolar or gitter-like appearance of the cell-structure with a contrary, shrunken and stumpy distorted appearance of the dendrites, and a more or less granular and opaque nucleus, are among the most notable changes.

In senile dementia, the cell shows a more uniform character, with less distortion, and only a fair fibrillary structure is preserved. The cell picture is generally darker, the fibrils are frequently matted, fragmentary or granular; the nucleus is often negative, or not infrequently more or less opaque and granular. The pigment masses are often notably increased in the smaller as well as the larger pyramids; their reticular net is often coarse and lumpy.

A cell-picture comparable to that of senile dementia is seen in the external geniculate in optic atrophy. Here the question of the plasmatic or fibrillary origin of the net of the pigment mass is not so clear.

In the fever change, naturally or artificially produced, present with the above change or alone, there is more or less apparent fragmentation and granulation of the fibrils corresponding to the Nissl-picture, as described by A. Hoch.

In central neuritis, described by Adolf Meyer in *Brain*, 1901, there is a parenchymatous alteration of the larger cell elements, mainly of central distribution appearing as a typical axonal reaction, with or without other superimposed cell changes. In the Beilschowsky equivalent a disappearance of the fibrils exactly corresponding to the area of swelling in the Nissl-equivalent. This is also shown by the harmony of the picture in the vortex type of this alteration.

The facts presented go to show the advantage of this new means of demonstrating histological details. On account of the variability in the Cajal picture in various parts of the same section and for ease of manipulation the Bielschowsky method has proved of especial advantage. A more intimate knowledge of the nervous structure and the finer histology of its components is promised through its wider use. The neuro-fibrillar structure appears to be quite as susceptible to morbid alterations as does the stainable substance of Nissl. Where the latter suffers the former shows equal changeability. There is abundant reason for reserve in assigning particular physiological attributes to the neuro-fibril.